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CASE REPORT

Kava-potentiated Withdrawal in Persons Using Multiple Substances: A Case Series of Kava and Kratom Co-ingestion

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Objectives: Kava (*Piper methysticum*) is indigenous to Pacific Island nations and has a long history of use in traditional medicine and ceremonies. Kava has effects similar to benzodiazepines, but its detailed mechanisms are uncertain. Although legal across the United States, kava is not without side effects. Currently, there are many kava and kratom blends accessible at stores and online. Recent case reports have emerged describing patient experiences with kava withdrawal when co-ingesting kava and kratom together; however, research remains scarce. This case series aims to add to the limited literature on kava withdrawal in co-ingestion of kava-kratom.

Methods: Publications before July 2025 were searched using the Ovid Medline, PubMed, and Google Scholar databases using the terms "kava", "kratom", "withdrawal", and "case report". The 2 case reports were found describing kava withdrawal symptoms and treatment when co-ingesting with kratom. This case series presents 2 additional cases from our institutions.

Results: All 4 patients were male with underlying histories of psychiatric illness and alcohol use disorder. All consumed more than the recommended amounts of the kava and kratom. Kava withdrawal symptoms were similar to sedative withdrawal. The onset of these symptoms varied considerably in relation to the last dose. Paradoxically, symptoms of kava withdrawal were observed in some cases despite sedatives still being present in the patients' systems; however, the involvement of polysubstance complexes the exact etiology of symptoms. All patients improved with phenobarbital and/or benzodiazepines.

Conclusions: Exact kava mechanism is complex and not fully understood. Further research is warranted.

Key Words: kava, feel free classic tonic, kava withdrawal, kratom.

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Kava *(Piper methysticum),* a plant indigenous to Pacific Island nations, has been used in traditional medicine, religious, and cultural ceremonies.[1,2] Kava has well-documented sedative/hypnotic effect[1] binding to $GABA_A$ similar to benzodiazepines;[2,3] however, there are reports of seizures in kava intoxication.[4] This paradoxical effect be possibly explained from kava's negative modulation of glycine receptors, as reduced function of these receptors is known to potentially trigger seizures; the exact mechanisms of kava remain unclear.[3,5,6] The half-life of kava is ~9 hours.[7] Kava, legal in all 50 states,[8] unfortunately has side effects such as hepatotoxicity and dermatologic reactions.[2,5,9] Kava can cause false-positive trigger for amphetamine immunoassay.[3] Recently, kava withdrawal has been reported when co-ingested with kratom.[3,10]

Many liquid blends of kratom and kava, such as Feel Free classic Tonic (FFCT) (Botanic Tonics, Broken Arrow, Oklahoma), Blue Razz (Mira9, Fort Myers, Florida), and KFusion (Zion Herbals, Richmond, Virginia) are available on the market and priced around $9.00 per bottle.[11–13] One bottle of FFCT contains 520 mg of kavalactones and dried leaf kratom (68 mg alkaloids).[11] Manufacturer recommends maximum one bottle daily; its effect can be experienced 10–15 minutes after consumption and can last 3–4 hours.[11] It is not recommended for those with history of substance misuse and warns habit forming nature due to kratom.[11]

Despite increased accessibility of kava and kratom blends, the research is scarce. Although withdrawal symptoms produced by kratom[14] (Fig. 1), and its treatment have been suggested in existing studies, the details of kava withdrawal and treatment, particularly when used in conjunction with kratom, are not well understood.[3,10] This case series aim to contribute to the limited body of knowledge on kava withdrawal when kava and kratom are co-ingested. Publications before July 2025 were searched using the Ovid Medline, PubMed and Google Scholar databases using the terms "kava" "kratom" "withdrawal" and "case report". Three case reports of co-ingestion of kava and kratom were found. One focused on kratom withdrawal, hence it was not included. The 2 case reports found and 2 additional cases from our institution

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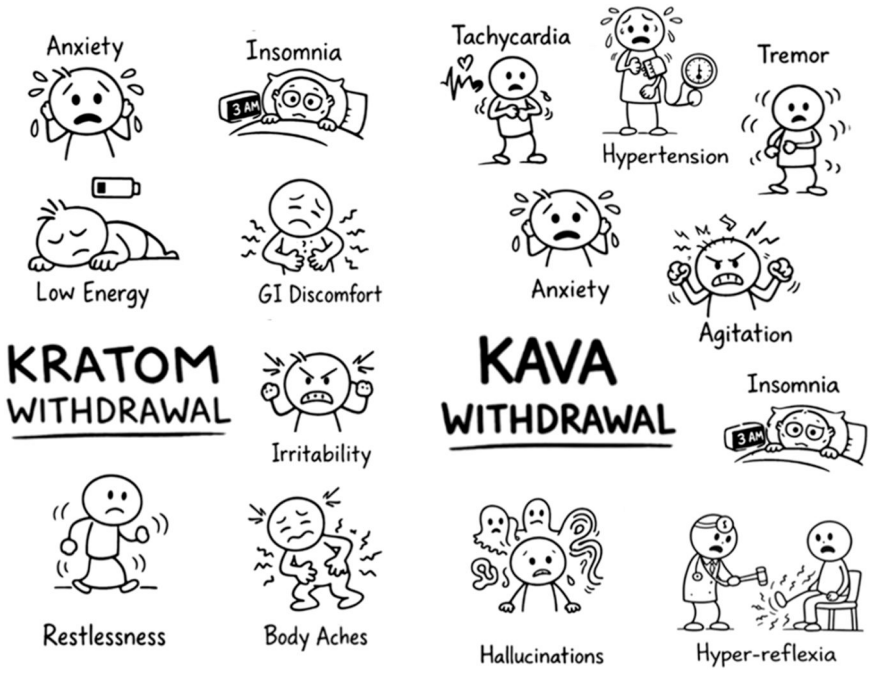

FIGURE 1. Common kratom and kava withdrawal symptoms. Picture produced with AI (ChatGPT) assistance, which was further edited by the author.

(consents for publications were obtained and were approved by the Institutional Review Board from our institution for both cases) are outlined in Table 1.

CASE PRESENTATION

Case A

Male in his mid-60s with major depressive disorder (MDD), alcohol use disorder (AUD), and chronic prescription diazepam 30 mg daily for general anxiety disorder (GAD) started consuming 1 bottle of FFCT daily, believing it was a healthier alternative to alcohol 1 year prior to admission (PTA). He was eventually consuming 15–18 bottles daily. Three days PTA, due to an ultimatum from the family, the patient stopped FFCT and diazepam. A night of admission, the patient felt hopeless and took diazepam 80 mg with FFCT. In the emergency department (ED), he was noted to be sleepy but was oriented except for a year. Brain computed tomography, labs, and vital signs were within normal limits. He was somnolent on day 2. On day 3, the patient had the first seizure-like activity followed by unresponsiveness for 1–2 minutes. He was appropriate and calm immediately after and the incident was not attributed to sedative withdrawal due to patient's age and recent diazepam overdose. Early afternoon, the patient had another seizure-like episode, foaming in the mouth with locking of joints. He was unresponsive for ~1 minute. He became more confused. Lorazepam 2 mg injection was given and oral diazepam 5 mg at bedtime was started. EEG was not done. Over the next 2 days, the patient had persistent borderline tachycardia and hyperactive delirium with hallucination. He required physical and chemical restraints. On day 6, the addiction medicine team was consulted and noted hyperreflexia. The team increased oral diazepam to 20 mg every 6 hours for suspected sedative (kava and diazepam) withdrawal. Next day, the patient's mentation improved significantly and diazepam tapering began. Restraints were discontinued. On day 9, he was back to his baseline. The patient and his family were educated about the danger of stopping sedatives abruptly. He was discharged on day 10.

Case B

A male in his 50s with AUD, MDD, and GAD transitioned from illicit opioid use to kratom 10 years PTA. Three years PTA, he transitioned from kratom to drinking 2 bottles of FFCT daily, which gradually increased to 12 bottles daily. He tried quitting FFCT but was unable to and also reported worsening of auditory hallucinations. Patient continued to consume 12 bottles of FFCT with one-fifth bottle of whiskey daily. Last FFCT consumption was one day PTA.

On the day of admission, he endorsed suicidal ideation, paranoia and visual hallucinations of demons. He was tachycardic, hypertensive, and exhibited minor leg tremors. His blood alcohol level (BAL) was 0.31 g/dL. His urine drug screen (UDS) was positive for amphetamine, which he denied using. He was placed on oral diazepam 10 mg every 6 hours and Clinical Institute of Withdrawal Assessment (CIWA) protocol. He was started on buprenorphine-naloxone and his home medication, quetiapine

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TABLE 1. Summary of Case Reports, Including 2 Cases From Our Institution

	Cassidy et al[3]	Bleifuss et al[10]	Case A	Case B
Sex	Male	Male	Male	Male
Age	30s	45	Mid-60s	50s
Chief complaint at emergency room presentation	Fall due to consuming too much kava	Auditory and visual hallucinations, anxiety, insomnia, and muscle jerking.	Suicide attempt: Diazepam overdose with FFCT ingestion	Suicide ideation
Psychiatric history	•Major depressive disorder •Attention deficit hyperactivity disorder	•Major depressive disorder •Obsessive compulsive disorder	•Major depressive disorder •Generalized anxiety disorder	•Major depressive disorder •Generalized anxiety disorder
Psychiatric hospitalization in the past	Had 3 psychiatric hospitalizations, each for depression and alcohol withdrawal.	NR	No	Multiple hospitalization due to alcohol withdrawal and suicidal ideation in the past
Outpatient psych meds	•Aripiprazole 10 mg nightly, •Fluoxetine 60 mg daily, •Buspirone 15 mg twice daily, •Gabapentin 300 mg 3 times daily.	•Fluvoxamine, •Buprenorphine 2 mg 3 times a day started by PCP for suspected kratom withdrawal 5 d PTA, which was increased to 4 mg 3 times a d 1 day PTA	•Diazepam 10 mg 3 times a day (per history, patient was taking average 20 mg daily) •lexapro 20 mg daily	•Quetiapine 200 mg at bedtime, •Divalproex ER 500 mg daily •Buprenorphine-Naloxone 4 mg–1 mg film daily (started 3 wk PTA)
Recent emergency room / hospital encounter	NR	Seen twice in emergency room over the past several months for suspected complications from FFCT—sedation, gait instability, and falls	Presented to another institution 5 d PTA asking to be detoxed from FFTC. He was not admitted.	Presented to another institution 3 wk PTA to be detoxed from FFTC. Patient was admitted and was sent home with buprenorphine 4 mg–1 mg daily.
History of alcohol use disorder (AUD) (severity of AUD)	Yes (severity NR), in remission	Yes (severity NR)	Yes (severe)	Yes (severe)
Recent alcohol use	No alcohol use for 5 mo PTA	No recent alcohol use	No recent alcohol use	Yes, binged drinking (2 bottles of 750 mL of Jack Daniel daily) for 5 d leading up to this admission. Serum alcohol level upon admission was 0.31 g/dL
Duration of use, amount and type of kava	•Started to take kava root extract, 3–4 mo PTA •1 mo PTA, consuming ½ lb bag daily (~20 servings) •Days PTA, several ½ lb bags daily •Patient reported long-standing daily consumption of 1/4 bag of kratom	•Duration of use, NR •FFCT, 8–10 bottles daily •Stopped 6 d PTA	•Started FFCT 1 y PTA •Started out with just 1 bottle a day, most recently drinking 18 bottles per day •Drank unknown amount on the day of admission with 80 mg of diazepam	•Started FFCT 3 y PTA •Started out with 2 bottles daily, most recently drinking 12 bottles per day with fifth bottle of Jack Daniel Whisky
Reasons for using kava	To suppress alcohol craving	NR	To quit / substitute for alcohol	To stay away from illicit opioid use
DSM-5 criteria fulfilled for "other substance" use disorder (kratom/kava)	Yes	Suspected of kratom/kava use disorder from history, symptom presentation and dermatological findings. The use of DSM-5 was not documented	Yes—used longer period than intended, unsuccessful efforts to cut down, tolerance, and withdrawal.	Yes—used longer period than intended, unsuccessful efforts to cut down, tolerance, and withdrawal.
Taking any other substance aside from kratom/ kava upon admission	Denied	NR	Diazepam 80 mg (took more than prescribed)	Alcohol and Seroquel 400 mg PTA (took more than prescribed)

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TABLE 1. (*continued*)

Onset of kava withdrawal symptoms	Unclear from the article: few hours to 12 h after admission Came in due to fall from intoxication of kava yet was agitated and required droperidol and midazolam to obtain imaging due to agitation upon admission suggesting possible kava withdrawal symptoms shortly after presenting to emergency room	Presented to emergency room 6 d after the last FFCT	Gradual onset starting hospital day 3	1 d after last FFTC
Summary of events leading up to hospitalization	•5 mo PTA: stopped alcohol •3–4 mo PTA: started kava root extract •1 mo PTA: consumed up to ½ lb kava daily (~20 servings) •Days PTA: consumed several bags of kava daily	•Unclear when FFCT was started, consuming 8–10 bottles daily •6 d PTA: stopped FFCT •5 d PTA: buprenorphine-naloxone 2 mg–0.5 mg 3 times daily was started for kratom withdrawal 1 d PTA: buprenorphine-naloxone was increased to 4 mg–1 mg 3 times a day	• > 5 y PTA: prescribed diazepam 30 mg daily (patient reported taking average 20 mg daily) •1 y PTA: started FFCT, 1 bottle daily •6 mo PTA: started to drink more FFCT and quit alcohol •4 mo PTA: started to drink alcohol again •3 mo PTA: quit FFCT but felt sick, continued to drink alcohol •6 wk PTA: started FFCT again drinking 4–8 bottles per day •20 d PTA: drinking up to 18 FFCT bottles daily •3 d PTA: Diazepam, FFCT and alcohol all stopped due to family ultimatum •Day of admission: felt sick and hopeless. Took 8 tablets of 10 mg diazepam + unknown amount of FFCT at night and came to emergency room	• > 20 y PTA: work injury, started to use illicit opioids •10 y PTA: stopped illicit opioids, started to use kratom, quantity not specified but described as using "several scoops" daily •3 y PTA: transitioned to FFCT, initially consuming 2 bottles daily •1.5 y PTA: FFCT consumption escalated to 12 bottles daily, he attempted to quit but wasn't able to and reported worsening of auditory hallucinations •3 wk PTA: attempted detox from FFCT at a local facility, buprenorphine-naloxone was started—a week later, resumed consuming 12 bottles of FFCT daily in combination with one-fifth bottle of Jack Daniel's whiskey daily •Day of admission: had suicidal ideation. Admits to drinking one-fifth bottle of Jack Daniel's whiskey. Last FFCT consumption was 1 d PTA. •IV lorazepam was administered by ambulance providers before arrival to the emergency department due to active hallucinations.
Kava withdrawal symptoms				
Vital signs	•Borderline tachycardia •BP 155/90 mmHg •RR 26/min •Afebrile	•Tachycardia •BP 144/128 mmHg •RR 20/min •Afebrile	•Borderline tachycardia •BP—with in normal limit •RR 18/min •Afebrile	•Tachycardia (130s/min) •BP 174/109 mmHg •RR 16/min •Afebrile
Hallucinations	No	Yes—auditory and visual	Yes—visual (seeing a guy with a gun)	Yes—tactile, auditory, and visual (people are talking about him, detectives are outside the room to get him, seeing / hearing demons)
Hyperactive delirium	Yes	No	Yes	No
Tremor	Yes	Yes	No	Yes
Agitation	Yes	NR	Yes	Yes
Anxiety	NR	Yes	Yes	Yes
Insomnia	Yes	Yes	Yes	Yes
Muscle jerking	Yes	Yes	No	No
Seizure-like activity	No	No	Yes (×2)	No
Hyper-reflexia	No	Yes (3+)	Yes (3+)	NR
Treatment and response to treatment				

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Treatment given for kratom withdrawal	No Buprenorphine-naloxone treatment, used full opioid agonist for kratom withdrawal	Buprenorphine-naloxone started as outpatient was continued	No buprenorphine or kratom withdrawal treatment (patient did not have any opioid withdrawal symptoms)	Buprenorphine-naloxone was started at 2-0.5 mg daily, which was adjusted and maintained at 8-2 mg twice a day by discharge from the hospital
Treatment given for kava withdrawal	6-d phenobarbital protocol •D 1: 260 mg, 360 mg, 360 mg IV, 3 h apart •D 2/3: 60 mg PO twice daily •D 4/5: 30 mg PO twice daily •D 6: 15 mg PO twice daily Continuous dexmedetomidine drip to hospital day 3 treated sympathetic activation and breakthrough agitation. For agitation: •Haloperidol 60 mg PO PRN (1st line) •Haloperidol 60 mg IV PRN (2nd line)	Phenobarbital 14.7 mg/kg (IV) ideal body weight given between day 1–3, then he was transitioned to diazepam PO 5 mg in d 3 and d 4.	Because of diazepam overdose, diazepam was initially held but due to seizure-like activities, diazepam 5 mg at bedtime was started on hospital day 3. •On hospital day 6, due to persistent hyperactive delirium and possible kava / diazepam withdrawal, diazepam was changed to 20 mg every 6 h •D 7: Diazepam was decreased to 10 mg every 6 h •D 8: Diazepam was decreased to 10 mg 3 times a day (outpatient prescription dose)	Patient was placed on diazepam 10 mg PO every 6 h with Clinical Institute Withdrawal Assessment (CIWA) protocol to keep CIWA score below 8 upon admission. •Diazepam tapering started hospital day 4. All benzodiazepines were tapered off before discharge. Quetiapine was increased from 200 mg at bedtime (outpatient dose) to 300 mg at bedtime due to persistent hallucination in d 2 and was discharged with this dose.
Mentation	improvement	2 d after the treatment (Hospital d 4)	Patient seemed not to have any mentation issue throughout the course	The day after the treatment
Hospital day 4				
Labs and other findings				
Hyponatremia	Yes—112 mEq/dL	Yes—133 mEq/dL	No	No
UDS	(+) for amphetamine – negative confirmatory test	NR	(+) for benzodiazepine (on prescription diazepam at home)	(+) for amphetamine but patient denied using
Kava dermopathy	No	Yes	No	No
Hospital course				
1st day	•Reported fall with transient loss of consciousness due to taking too much kava. •Agitated and required droperidol and midazolam to obtain imaging •Required multiple medications including IV hydromorphone, droperidol, haloperidol, midazolam as well as po hydrocodone and acetaminophen	•Reporting auditory and visual hallucinations, anxiety, insomnia and muscle jerking •Tachycardic and hypertensive •Pressured speech, tremor, 1–2 beats of clonus with 3+ patella reflexes •Intravenous phenobarbital 10 mg/kg ideal body weight was given which led to significant improvement in symptoms but hyper-reflexia persisted and had difficulty relaxing his legs •Cyproheptadine 12 mg po given but no significant improvement •Buprenorphine-naloxone outpatient dose was continued	•Slow response but able to answer all questions. •Computed Tomography head was within normal limits; lab tests were normal	•Tachycardic, hypertensive, tremors, serum alcohol level was 0.31 g/dL. •Started on diazepam 10 mg every 6 h in addition to CIWA protocol. •All home medications restarted •Buprenorphine-naloxone were restarted at 2–0.5 mg with dose adjustment
2nd day	•Psychiatry consulted for agitation and withdrawal (12 h after admission) •Not oriented to month with fluctuating attention with substantial distractibility, very poor short-term memory with agitation, no hallucinations •Diffuse fine tremors, normal reflexes •Borderline tachycardia, tachypnea and hypertension •As needed haloperidol 60 mg po (1st line)	•2 doses of IV phenobarbital 130 mg given •Clonidine 0.1 mg po 3 times a day started	•Somnolent •Outpatient diazepam was held •Oriented to self and place only	•Continue to have auditory hallucinations and paranoia •Quetiapine dose was increased from 200 to 300 mg •Multiple diazepam 10 mg were required to keep CIWA less than 8

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TABLE 1. (*continued*)

	or IV (2nd line) for agitation •Dexmedetomidine drip started in addition to 6-d phenobarbital protocol •6-d phenobarbital protocol d #1: IV phenobarbital slow push of 260 mg, 3 h later 360 mg, then 3 h later 360 mg			
3rd day	•Phenobarbital protocol d #2: 60 mg po phenobarbital twice daily •Dexmedetomidine drip discontinued	•1 dose of IV phenobarbital 130 mg given —total 14.7 mg/kg •Diazepam 5 mg po given	•Seizure-like activity × 2, •After 2nd seizure-like activity, he was responding "2132" when asked where he was •Lorazepam 2 mg IV was given for seizure and diazepam 5 mg po at bedtime started	•Continued to have auditory hallucinations, however rest of the withdrawal symptoms started to improve •CIWA protocol continued
4th day	•Phenobarbital protocol d #3: 60 mg po phenobarbital twice daily •Fully oriented with intact memory, calm, cooperative, no hallucinations	•Clonidine discontinued •Diazepam 5 mg po given •Discharged home	Severely agitated, yelling and screaming requiring intravenous haloperidol 1 mg and lorazepam 0.5 mg.	•Hallucinations resolved. •Diazepam taper started
5th day	•Phenobarbital protocol d #4: phenobarbital 30 mg po twice daily •Cleared for discharge with remainder of phenobarbital protocol •Kratom withdrawal was treated with full opioid agonist (oral oxycodone and intravenous hydromorphone) during admission	NA	•Actively hallucinating and yelling. •Olanzapine 2.5 mg and lorazepam 1 mg per oral given •Patient was on physical restraints for safety	•Denied suicidal ideation. •Diazepam taper continued
6th day	Phenobarbital protocol d #5: phenobarbital 30 mg po twice daily	Followed up as outpatient—reported sleeping well and no hallucinations, opioid/sedative withdrawal symptoms	•Addiction team consulted •For possible persistent hyperactive delirium and sedative (benzodiazepines and kava) withdrawal, diazepam 5 mg po at bedtime was changed to 20 mg every 6 h	Buprenorphine-naloxone, which was adjusted since admission was now stable at 8 mg-2 mg twice daily
7th day	Phenobarbital protocol d #6: phenobarbital 15 mg po twice daily	•NA	•Calm and sleepy, now oriented to name, place, year and month •Diazepam was changed to 10 mg every 6 h	•Off diazepam •Discharged with his home medications, including buprenorphine-naloxone 8–2 mg twice a day.
8th day	•NA	•NA	•Physical restraints completely discontinued •Patient continue to improve •Diazepam was changed back to outpatient prescription dose, 10 mg po 3 times a day	•NA
9th day	•NA	•NA	Confirmed by patient's family that patient is completely back to his baseline	•NA
10th day	•NA	•NA	Discharged home	•NA

BP indicates blood pressure; FFCT, Feel Free Classic Tonic; HR, heart rate; IV, intravenous; NA, not applicable; NR, not recorded; PO, per oral; PTA, prior to admission; RR, respiratory rate.

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200 mg orally at bedtime. On day 2, the patient had persistent paranoia and hallucinations, hence, quetiapine was increased to 300 mg. He required multiple diazepam to keep CIWA below 8. On day 4, all hallucinations resolved and suicidal ideation improved. Diazepam tapering was started. On day 7, the patient was discharged home with buprenorphine-naloxone 16 mg–4 mg daily and quetiapine 300 mg at bedtime.

DISCUSSION

History

All 4 patients were male with age ranging between 30s and 60s and had a history of underlying MDD and AUD. At least 3 cases, the patients started co-ingesting kratom and kava in an attempt to get off or substitute the substance. Three patients were consuming FFCT, whereas one co-ingested separate products of kratom and kava together. All developed tolerance quickly and consumed above the manufacturer recommendation.[3,10] Educating patients about the risk of discontinuing sedatives, such as FFCT is important as seen in Bleifuss and colleagues, and case A. Patients may have the perception that kava is safe as it is a legal plant product.

Withdrawal Symptoms and Treatment

From all cases, kava withdrawal symptoms (Fig. 1) were similar to sedative withdrawal; agitation seemed to be a common denominator with 3 patients suffering from tactile, visual, and/or auditory hallucinations, 2 patients with hyperactive delirium, and 1 with seizure-like activities. Many had autonomic hyperactivity such as hypertension and tachycardia. Because of common involvement of $GABA_A$ by alcohol, benzodiazepines, and kava, despite polysubstance use in case A and B, as in all 4 cases, symptoms resolved after treatment with sedatives, such as phenobarbital and/or diazepam.

Withdrawal Timeline

Timeline of the withdrawal seems to be inconsistent in all cases, which may be due to multiple factors including difference in strength of kava consumed, one's tolerance, and quantity/duration of use. In Cassidy and colleagues, the patient was admitted for kava intoxication, however, required droperidol and midazolam to obtain imaging due to agitation upon admission. In Bleifuss and colleagues, use of opioids (buprenorphine-naloxone) may have possibly masked initial minor kava withdrawal symptoms (ie, anxiety),[15] which led to ED 6 days after discontinuation of FFCT. In case A and B, kava withdrawal-like symptoms were observed despite significant sedatives in their systems. In case A, the patient overdosed on diazepam, which has a half-life of 46 to 100 hours.[16] Initial sedative withdrawal-like symptoms occurred on day 3; however, this was not attributed to sedative withdrawal due to the long half-life of diazepam in the elderly.[17] In case B, pt had a serum alcohol level of 0.31 g/dL, yet, he had sedative withdrawal-like symptoms. Although polysubstance use limits the exact etiology as symptoms may be attributed in case A to co-ingested diazepam withdrawal and in case B, early withdrawal from severe alcohol use and/or possible amphetamine use, inconsistency in kava withdrawal timeline among the 4 patients warrants further research, especially when co-ingested with kratom.

Common Labs

Both Cassidy and Bleifuss and colleagues, the patient had hyponatremia. It is unclear if this is a direct or indirect effect of kava. In Cassidy and colleagues, and case B (confirmatory test not ordered), patients had false-positive for amphetamine, which was attributed to kava.

CONCLUSIONS

Although kava is easily accessible, research is scarce, especially co-ingestion with kratom. From the 4 cases reviewed, there are some commonalities, and there are still unknowns, especially withdrawal timeline. Because of its legality, people may not perceive kava as a "drug"; clinicians therefore need to ask specifically about "health supplement" or "substance over the counter", which may lead to early intervention, education and prevention, especially further complications, such as kava withdrawal. As evident in case A and B, polysubstance use significantly complicates the clinical picture of kava withdrawal symptoms and management. This case series further adds to the paucity of literature describing the adverse consequences associated with sudden kava cessation when co-ingested with kratom.

Limitation

In our cases, other substances that are not routinely tested on UDS may have contributed to their symptoms. In addition, their underlying mental illness, polysubstance use and medical problems may have contributed to hallucinations / delirium. Their mental status improved with diazepam, suggesting symptoms were from sedative withdrawal; however, due to polysubstance use, we couldn't definitively state that symptoms were from kava withdrawal alone.

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